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ORIGINAL ARTICLE

Evaluation of the risk factors and clinical features of newly developing cerebral microbleeds following internal carotid artery stenting

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ABSTRACT

Objectives: This study aimed to characterize the clinical features of incident cerebral microbleeds (CMBs) following internal carotid artery (ICA) stenting and identify related risk factors.

Patients and methods: This retrospective observational study included 80 patients who underwent ICA stenting between January 2021 and June 2021. All patients underwent cranial magnetic resonance imaging (MRI), diffusion MRI, arterial spin labelling perfusion MRI, and susceptibility-weighted imaging, which were performed before the ICA stenting procedure and 72 h after the procedure. Risk factors associated with CMBs were analyzed by comparing patients who developed new CMBs following ICA stenting with those who did not.

Results: The mean age of the patients was 64.95 ± 11.14 years. Fourteen patients (17.50%) were female, and 66 (82.50%) were male. New CMBs were detected in 25% of the patients following the ICA stenting procedure. A statistically significant association was found between the development of new CMBs and the presence of symptomatic ICA stenosis and anemia but not with any of the other parameters.

Conclusion: Considering the development of new CMBs as a potential predictor of intracranial hemorrhage may facilitate closer monitoring and the adoption of personalized treatment strategies for this patient population.

Keywords: Anemia, carotid artery stenting, cerebral microembolism, cerebral microhemorrhages.

The management of internal carotid artery (ICA) stenosis is crucial for preventing and treating ischemic stroke.[1] Internal carotid artery stenosis is a major risk factor for ischemic stroke, with the annual incidence of stroke in symptomatic cases reaching up to 26%.[2-4] Although ICA endarterectomy has long been regarded as the standard treatment for ICA stenosis, the adoption of ICA stenting has markedly increased worldwide in recent years.[5,6] The findings of a comparative study of ICA stenting and ICA endarterectomy indicated that ICA stenting may be as safe as ICA endarterectomy, particularly in younger patients.[7] Another study showed that ICA stenting was associated with an increased risk of early periprocedural stroke, although the long-term outcomes of ICA stenting and ICA endarterectomy were comparable.[8] These findings indicate that both ICA stenting and ICA endarterectomy can be safely performed in specific patient populations. The most critical complications following ICA stenting include stroke, restenosis, hyperperfusion syndrome, and hemorrhagic changes.[9] Recently, cerebral microbleeds (CMBs) have emerged as a valuable predictor of risk of intracranial hemorrhage. Several studies have

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established a strong association between CMBs and the likelihood of subsequent intracranial hemorrhage.[10-13] Histopathological analyses have revealed that CMBs may be precursors to acute hemorrhage.[10-13] Histologically, CMBs appear as small perivascular deposits of hemosiderin-laden macrophages; the hemosiderin is formed following the phagocytosis of erythrocytes extravasated from small cerebral vessels. On magnetic resonance imaging (MRI), particularly on T2-weighted gradient-echo (GRE) and susceptibility-weighted imaging (SWI) sequences, CMBs appear as small, round, hypointense lesions measuring 5 to 10 mm in diameter.[10,11] Cerebral microbleeds are classified into two categories: hypertensive vasculopathy and cerebral angiopathy.[12] These two categories are associated with hypertension (HT), advanced age, male sex, and low cholesterol levels.[13] Only a few studies in the literature have examined the risk factors associated with the development of CMBs following ICA stenting during the early follow-up period by directly comparing pre- and postprocedural MRI scans. Therefore, early detection of incident CMBs and identification of related risk factors in patients undergoing ICA stenting are of significant clinical importance. Given the limited evidence on newly developed CMBs after ICA stenting, further studies are needed to clarify their clinical characteristics and associated risk factors in this specific patient group. Hence, this study aimed to characterize the clinical features of incident CMBs in this patient population and identify the associated risk factors.

PATIENTS AND METHODS

This retrospective study included 80 patients who underwent ICA stenting at the Neurology Outpatient Clinic of the Uludağ University Faculty of Medicine, between January 2021 and June 2021 (Figure 1). The inclusion criteria were as follows: ICA stenosis of 70% or greater confirmed by computed tomography (CT) angiography, indication for ICA stenting, cranial magnetic resonance imaging, including SWI and arterial spin labelling (ASL) perfusion, performed 24 h before and 72 h after the ICA stenting procedure, regular follow-up, and cranial neck CT angiography to assess stent placement at the one-year follow-up. The exclusion criteria included the following: discontinuation of neurology follow-up, presence of atrial fibrillation, and renal failure. Written informed consent was obtained from all participants. The study protocol was approved by the Uludağ University Faculty of Medicine Ethics Committee (Date: 22.05.2024, No. 2024-8/4). The study was conducted in accordance with the principles of the Declaration of Helsinki.

The patient demographics (age and sex), vascular risk factors (HT, diabetes mellitus [DM], and hyperlipidemia), and current medication use were recorded. Computed tomography angiography was performed with a 128-slice Somatom Definition AS+ CT scanner (Siemens Healthineers, Erlangen, Germany). Noncontrast and contrast-enhanced axial images spanning the aortic arch to the vertex were obtained, and the degree of stenosis and stent patency were assessed using coronal reconstructions. The patients received

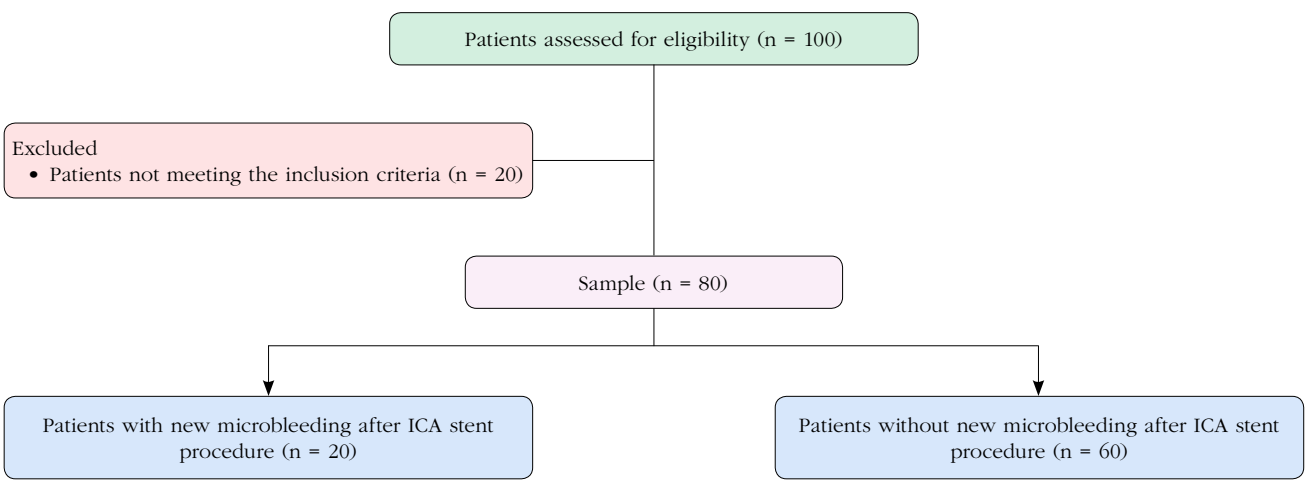

Figure 1. The flowchart shows patients who underwent the ICA stent procedure, including those in the study.
ICA, internal carotid artery.

dual antiplatelet therapy with acetylsalicylic acid (100 mg/day) and clopidogrel (75 mg/day) before stenting. All stenting procedures were performed using a transfemoral catheterization technique with the patient under local anesthesia. Intravenous heparin was administered during the procedure, and a distal embolism protection filter device (FilterWire EZ; Boston Scientific, Marlborough, MA, USA) was used in all patients. The placement of a Boston Wallstent (Boston Scientific, Marlborough, MA, USA) was preceded by predilation when necessary. Postdilation was performed using a 5.0- to 6.0-mm balloon suited to the diameter of the ICA distal to the stenotic segment, and it was held at 6 to 10 atm for 10 to 30 sec. All patients were subsequently monitored in the neurology department for at least 72 h after the procedure, and the findings of hourly neurological examinations were recorded during the first 24 h, followed by routine neurological monitoring thereafter. All patients underwent cranial MRI on a MAGNETOM Aera 1.5 Tesla MRI scanner (Siemens Healthineers, Erlangen, Germany) 24 h before and 72 h after the procedure. The protocol included SWI, T1-weighted imaging, T2-weighted imaging, FLAIR (Fluid-attenuated inversion recovery) imaging, diffusion-weighted imaging, and ASL perfusion sequences (three-dimensional pulsed ASL with inversion times based on the International Society for Magnetic Resonance in Medicine recommendations). To assess CMBs, SWI sequences were evaluated by two experienced neuroradiologists (23 and seven years of experience) who were blinded to the clinical and procedural data. Cerebral microbleeds were defined as round or ovoid hypointense foci measuring < 10 mm in diameter on SWI. Vascular flow voids, calcifications, and artefacts were excluded. Incident CMBs were recorded by comparing pre- and postprocedural SWI images. Arterial spin labeling images were used to assess perfusion and were qualitatively examined using automatically generated perfusion maps. A color look-up table was used in all cases. Cortical grey matter showed a signal below the expected level, indicating hypoperfusion. As shown in Figures 2 and 3, the presence of arterial transit artefacts was evaluated in favor of hypoperfusion. To assess the presence of hyperperfusion, postprocedural images were compared to preprocedural images, and incident hyperintense cortical areas with increased signal relative to the contralateral hemisphere were defined as hyperperfusion. Concurrently, new

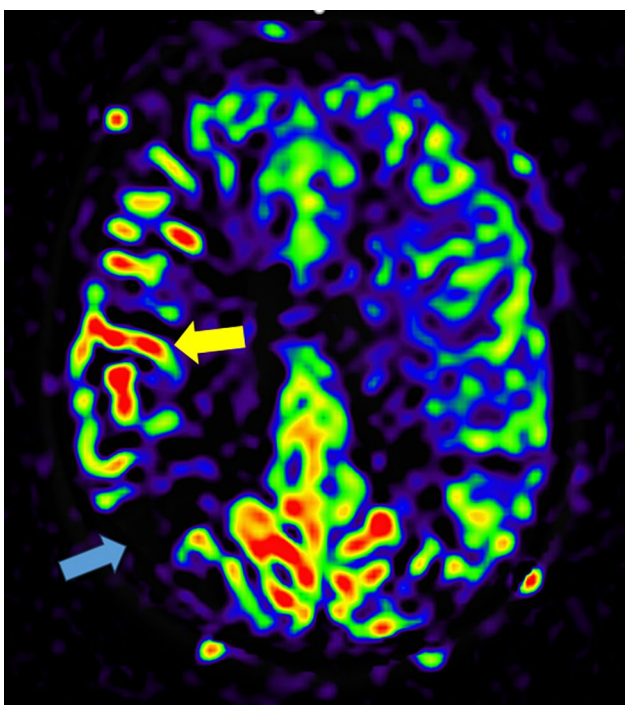

Figure 2. Cortical hypoperfusion (blue arrow) and arterial transit artifacts (yellow arrow) in the right frontoparietal region.

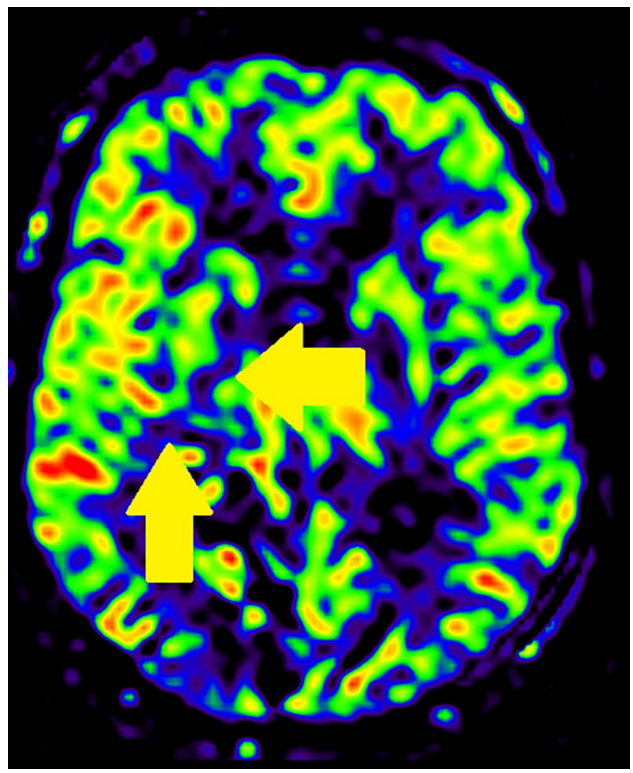

Figure 3. Ipsilateral right cerebral hemisphere hyperperfusion after stenting in a patient who underwent stenting for approximately 90% stenosis in the right ICA.
ICA, internal carotid artery.

CMBs detected on postprocedural SWI and incident acute infarct areas identified on postprocedural diffusion MRI were examined (Figures 4, 5). Leukoaraiosis was assessed using the Fazekas score on preprocedural FLAIR sequences.[14] The craniocervical atherosclerotic burden score was calculated using preprocedural CT angiography.[15] All patients were followed up in the neurology outpatient clinic for one year. At the end of the first year, patients were evaluated for stent restenosis and major bleeding events. Major bleeding was defined according to the International Society on Thrombosis and Hemostasis criteria as life-threatening extracranial or intracranial hemorrhage requiring hospitalization, blood transfusion, or surgical intervention or resulting in significant neurological deterioration or death.[16] Risk factors for CMBs were identified by comparing patients with incident CMBs to those without incident CMBs.

Statistical analysis

The clinical properties, demographic data, and radiological findings of the study participants were compared. All data analyses were performed using IBM SPSS version 23.0 software (IBM Corp., Armonk, NY, USA). The mean and standard deviation of age were calculated, and the following

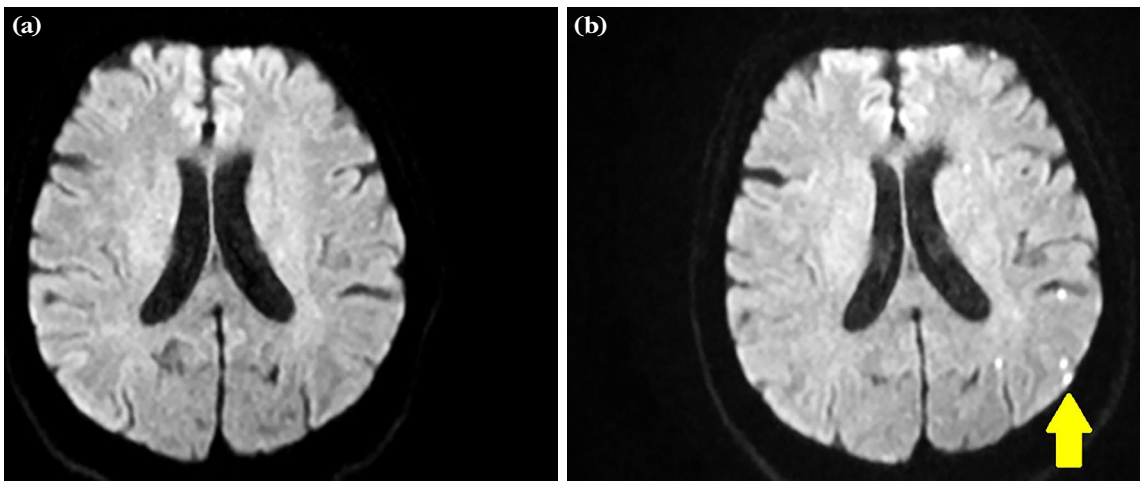

Figure 4. Infarct foci developing after the procedure in a patient who underwent stenting for 85% stenosis observed in the left ICA. **(a)** before stenting, **(b)** after stenting.
ICA, internal carotid artery.

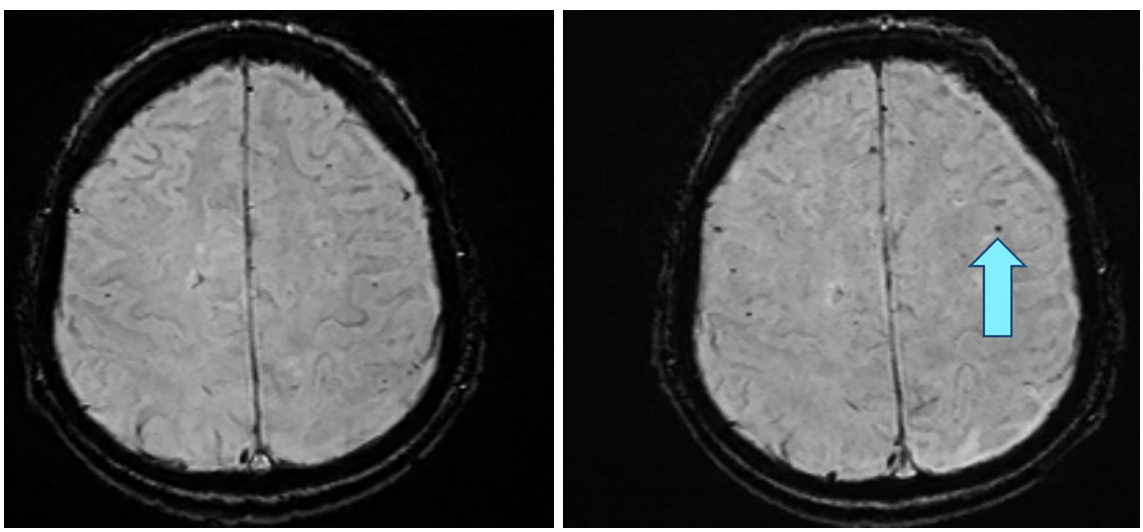

Figure 5. New CMB (blue arrow) after stenting in a case with left ICA stenting.
CMB, cerebral microbleed; ICA, internal carotid artery.

variables were also evaluated: sex, DM, HT, symptomatic ICA, hypoperfusion detected on preprocedural ASL perfusion MRI, CMBs detected on preprocedural SWI, new diffusion restriction detected on postprocedural diffusion MRI, new CMBs identified on postprocedural SWI MRI, hypoperfusion findings on postprocedural ASL perfusion MRI, early neurological deterioration, history of major bleeding within one year, and restenosis within the first year of stent follow-up. For continuous variables with normal distributions, the number, mean, and standard deviation were calculated, and an independent-samples t-test was used. For variables with nonnormal distribution, the median and the 25th and 75th percentiles were used, and the Mann-Whitney U test was applied. Categorical variables were expressed as percentages and analyzed using the chi-square test. A logistic regression model was developed using statistically significant variables (being symptomatic of the ICA and hemoglobin value) and potential confounding effects (craniocervical atherosclerotic burden score, age, and sex). A p-value < 0.05 was considered statistically significant.

TABLE 1
Evaluation of risk factors for newly developing CMBs after ICA stenting

	Patients with new CMBs after ICA stenting (n = 20)			Patients without new CMBs after ICA stenting (n = 60)			
	n	%	Mean ± SD	n	%	Mean ± SD	*p*
Age (year)			63.60 ± 9.38			64.40 ± 11.71	0.217*
Sex							
Male	14	70		45	75		0.774**
Presence of DM	10	50		31	51.66		0.897**
Presence of HT	16	80		42	70		0.563**
Leukoaraiosis	8	40		34	56.66		0.301**
Craniocervical atherosclerosis burden score			5.750 ± 3.552			4.833 ± 2.300	0.302*
Hemoglobin (g/dL)			11.905 ± 2.325			14.396 ± 9.689	0.030*
Creatine (mg/dL)			0.962 ± 0.278			0.932 ± 0.301	0.494*
LDL (mg/dL)			117.400 ± 40.012			116.480 ± 43.471	0.929*
HbA1c (%)			6.590 ± 1.685			7.346 ± 6.537	0.644*
Glucose (mg/dL)			151.200 ± 51.275			135.290 ± 64.010	0.173*
Symptomatic ICA stenosis	18	90		41	68.33		0.041**
Having more than 50% stenosis in the contralateral ICA	7	35		24	40		0.895**
Occlusion of the contralateral ICA	0	0		3	5		0.734**
Stenosis degree of ICA with stented			84.40 ± 21.13			87.45 ± 9.61	0.995*
Stenosis degree of contralateral ICA			28.25 ± 37.67			31.83 ± 40.60	0.744*
Hypoperfusion findings on ASL perfusion MRI before the procedure	12	60		27	45		0.366**
Presence of microhemorrhages on pre-procedure SWI MRI	7	35		19	31.66		0.784**
New diffusion restriction in post-procedure diffusion MRI	7	35		13	21.66		0.244**
Findings of hyperperfusion in ASL perfusion MRI after the procedure	2	10		2	3.33		0.554**
Early neurological deteriortion	0	0		2	3.33		0.747**
History of major bleeding within first year	0	0		2	3.33		0.833**

CMBs, cerebral microbleeds; ICA, internal carotid artery; SD, standard deviation; DM, diabetes mellitus; HT, hypertension; LDL, low-density lipoprotein; HbA1c, Hemoglobin A1c; MRI, magnetic resonance imaging; SWI, susceptibility weighted imaging; ASL, arterial spin labeling; * Mann-Whitney U test; ** Pearson chi-square/continuity correction/Fisher's exact test.

TABLE 2
Binary logistic regression evaluation of significant risk factors for newly occurring CMBs after ICA stenting

Variables	*p*	Odds Ratio	95% CI for Exp (B) Lower	Upper
Sex	0.234	2.544	0.547	11.833
Age	**0.006**	0.877	0.799	0.962
Hemoglobin level (g/dL)	**0.008***	0.636	0.454	0.890
Symptomatic ICA stenosis	**0.030****	11.351	1.274	101.168
Craniocervical atherosclerosis burden score	**0.009****	1.423	1.090	1.857

CMBs, cerebral microbleeds; ICA, internal carotid artery; CI, confidence interval. Significance of the model < 0.001. Significant variables are shown in bold.

RESULTS

The mean age of the patients was 64.95 ± 11.14 years. Fourteen patients (17.50%) were female, and 66 (82.50%) were male. Internal carotid artery stenosis was symptomatic in 59 patients (73.75%). The contralateral ICA showed > 50% stenosis in 31 patients (38.75%) and was occluded in three patients (3.75%). Thirty-nine patients (48.75%) showed signs of hypoperfusion on ASL perfusion MRI prior to ICA stenting. Twenty-six patients (32.50%) had CMBs detected on SWI performed before the procedure. After the procedure, 20 patients (25%) developed new CMBs on SWI, and 20 patients (25%) developed new diffusion restrictions on diffusion MRI. All new cerebral microhemorrhages detected after ICA stenting were ipsilateral, and their number varied. Predilation with balloon angioplasty before stent deployment was performed in 33 patients (41.25%). Postprocedural ASL perfusion MRI detected hyperperfusion in four patients (5%). Neurological deterioration occurred in two patients (2.50%) after the procedure, and two patients (2.50%) had a history of major bleeding. Stent restenosis was detected in two patients (2.50%) within the first year following the stenting procedure. Patients with incident CMBs following ICA stenting were compared with those without incident CMBs. A significant statistical association was found between incident CMBs following ICA stenting and symptomatic stenosis in the ICA in which the stent was placed (p = 0.041), as well as with hemoglobin levels (p = 0.030). Conversely, no significant association was observed with the following parameters: age (p = 0.217), sex (p = 0.774), DM (p = 0.897), HT (p = 0.563), leukoaraiosis (p = 0.301), contralateral ICA stenosis > 50% (p = 0.895), contralateral ICA occlusion (p = 0.734), degree of stenosis in the contralateral ICA (p = 0.744), preprocedural hypoperfusion findings (p = 0.366), detection of CMB on preprocedural SWI (p = 0.784), postprocedural diffusion restriction (p = 0.244), detection of hyperperfusion on ASL perfusion MRI (p = 0.554), craniocervical atherosclerotic burden score (p = 0.302), history of major bleeding within one year (p = 0.833), and stent restenosis within one year (p = 0.766; Table 1).

Binary logistic regression analysis was performed with significant variables (symptomatic ICA stenosis and hemoglobin levels) and potential confounding effects (age, sex, and cervicocerebral atherosclerosis burden score) for newly occurring CMBs after ICA stenting. According to our binary logistic regression results, the most significant variables were age (p = 0.024, odds ratio [OR] = 0.930), serum hemoglobin level (p = 0.012, OR = 0.669), symptomatic ICA stenosis (p = 0.022, OR = 11.534), and cervicocerebral atherosclerosis burden score (p = 0.022, OR = 1.360) (Table 2).

DISCUSSION

In this study, pre- and postprocedural MRI findings in patients who underwent ICA stenting were examined. Cerebral hemodynamic changes were assessed using SWI and ASL perfusion MRI. We found that 25% of the patients developed new CMBs after the ICA stenting procedure, and new diffusion restrictions were detected in 25% of the patients. Hyperperfusion was observed in 5% of the patients, and early neurological deterioration occurred in 2.5%. Although a significant association was found between the development of new CMBs and symptomatic ICA stenosis, no notable association was observed between incident CMBs and other clinical or procedural factors. Cerebral microemboli and new ischemic lesions following ICA stenting are widely documented in the literature.[17,18] However, the

significance of CMBs during the periprocedural period and the associated risk factors remain poorly understood. Nishikawa et al.[19] reported that new CMBs may be detected on SWI after ICA stenting and may be related to antithrombotic therapy and hemodynamic changes. Our findings support these observations by demonstrating the development of new CMBs after stenting. The link between symptomatic ICA stenosis and incident CMB suggests that this patient population may be particularly vulnerable at the microvessel level. In two previous studies that employ GRE sequences, the prevalence of CMBs after ICA stenting ranged from 8% to 10.4%.[20,21] In our study, the prevalence was 25%, which exceeds the figures reported in the existing literature. This discrepancy might be due to the use of SWI, which is recognized for its higher sensitivity compared to the GRE sequence. Because no studies incorporating SWI were available in the literature, we could not make a direct comparison with the results of other studies. Previous research has proposed that initial CMBs may signal the development of new microbleeds and that hemodynamic changes during ICA stenting could potentially harm small cerebral vessels.[21,22] Cerebral ischemic lesions have been observed following cerebral angiography and neurointerventional procedures, with cerebral embolic lesions detected on diffusion MRI in approximately 40 to 50% of patients who underwent ICA stenting with a protective device.[23,24] The pathophysiology underlying these emboli is varied, involving mechanisms such as plaque rupture and artery-to-artery embolism. In addition, increased wall shear stress in the ICA and its branches in patients undergoing ICA stenting can activate the von Willebrand factor, leading to thromboembolism via activation of the coagulation cascade.[6]

In this study, we observed new diffusion restrictions in 25% of patients after ICA stenting, which is lower than the rates reported in the literature. Interestingly, we found no significant statistical correlation between CMBs and new diffusion restrictions. Potential explanations include increased operator experience, routine use of ICA filters, and utilization of closed-cell stents. Furthermore, preprocedural ASL perfusion MRI detected signs of hypoperfusion in 39 patients, indicating limited hemodynamic reserve prior to stenting. Although only four patients (5%) in our study developed hyperperfusion after the ICA stenting procedure, this finding supports the occurrence of hyperperfusion syndrome, a clinically rare but potentially severe complication, following ICA stenting.[25] This low incidence may be linked to meticulous patient selection and optimal periprocedural management. In addition, no significant relationship was found between leukoaraiosis and the development of CMBs. Leukoaraiosis, as an indicator of small vessel disease, demonstrates microvessel fragility and diminished cerebral reserve.[26] Therefore, it has been proposed that leukoaraiosis may be a risk factor for microbleeds owing to hemodynamic fluctuations during stenting. Nonetheless, prior studies have produced inconsistent results, with some indicating that only the initial CMB burden predicts the development of new lesions and that leukoaraiosis is not an independent predictor.[27,28] The present study demonstrates that in patients undergoing ICA stenting, the total cervicocerebral atherosclerotic burden score was significantly associated with the development of new CMBs after the procedure. This association suggests that widespread atherosclerotic disease may reflect not only large vessel pathology but also cerebral microvascular fragility. A high atherosclerotic burden is associated with chronic endothelial dysfunction, arterial stiffening, and hemodynamic instability in the distal microcirculation, which can impair the structural integrity of cerebral small vessels and increase the sensitivity of the blood-brain barrier. Sudden hemodynamic changes following carotid revascularization can trigger new microbleed formation, particularly by leading to reperfusion stress in the predamaged microvascular bed. This mechanism suggests that CMBs observed after carotid stenting cannot be explained solely by procedural microembolism, and that the patient's underlying vascular burden and microvascular sensitivity may be important determinants. Therefore, the total cervicocerebral atherosclerotic burden score could be used as a potential imaging biomarker to predict the risk of postoperative hemorrhagic microvascular complications in patients scheduled for carotid stenting.[9,29] Furthermore, no significant association was observed between preprocedural predilation and the development of new microbleeds. Theoretically, predilation might heighten the risk of cerebral complications by inducing plaque rupture and distal embolism.[30] Some studies have reported a connection between predilation and new diffusion restrictions;[31] however, evidence linking predilation to microbleeds remains limited. Our results indicate that predilation was not a decisive factor in the development of microbleeds. Another significant

finding was the link between blood hemoglobin levels and incident CMBs in patients who underwent ICA stenting. A statistically significant association between low hemoglobin levels and the appearance of new CMBs following the procedure was demonstrated. Although previous studies have reported a correlation between anemia and CMBs in the general population, no prior research has specifically examined this relationship in the context of ICA stenting. To the best of our knowledge, this is the first study to investigate and establish such an association. Mechanistically, reduced hemoglobin levels may impair cerebrovascular resistance and cerebral blood flow by decreasing the oxygen-carrying capacity of the blood, thereby promoting hypoxia-induced endothelial dysfunction and microvascular fragility, ultimately facilitating the development of CMBs.[11,32-37]

The mechanisms underlying newly developed CMBs after ICA stenting are likely multifactorial and cannot be explained solely by procedural embolism. In addition to microembolic phenomena, abrupt hemodynamic alterations following carotid revascularization may contribute to disruption of vulnerable cerebral small vessels and blood-brain barrier instability. Patients with chronic hypoperfusion, diffuse atherosclerotic disease, or impaired cerebrovascular autoregulation may be particularly susceptible to these microvascular changes. Therefore, incident CMBs observed after ICA stenting may reflect both procedure-related embolic injury and underlying cerebral microvascular fragility.

This study had several limitations. First, the single-center design and relatively small patient population limit the generalizability of the results. Furthermore, the clinical implications of the CMBs detected on SWI could not be assessed through long-term follow-up. However, the notable strengths of this study include the use of the same imaging protocol before and after the procedure and the simultaneous examination of both CMBs and perfusion changes.

In conclusion, new CMBs developed in 25% of patients following ICA stenting and were more common in symptomatic cases. The development of CMBs following ICA stenting warrants clinical attention. In addition, findings of preprocedural perfusion restriction and occasional hyperperfusion indicate that cerebral hemodynamics should be carefully monitored in this patient population. Larger patient series and long-term follow-up studies are needed to further clarify the clinical implications of these findings.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Y.D., B.H.: Idea/concept; Y.D., B.H.,R.Ö.: Design; B.H., M.B., R.Ö.: Control/supervision, critical review; G.G., F.S., E.O.A.: Data collection and/or processing, literature rewiew; Y.D., G.G., R.Ö.: Analyses and/or interpretation, references and fundings; Y.D., B.H., R.Ö.: Writing the article, materials.

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